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ORIGINAL ARTICLES.

DENDRITIC KERATITIS.

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THE clinical picture of dendritic, or "malarial," keratitis may best be exhibited by the following extracts from those authors who have first called our attention to this form of corneal disease:

KIPP (*Trans. Am. Ophth.*, 1880, p. 91) says: "Dendritic keratitis commonly developed within a few days after an attack of intermittent fever, often simultaneously with the appearance of herpetic vesicles on the nose or lips. In some cases every fresh attack of the fever was followed by this corneal affection." Usually only one eye is affected. "Shortly after the first symptoms of irritation, one, or two or more, slightly raised, irregular, opaque lines of varying length will be found on different parts of the cornea. At the same time some circum-corneal injection will be present. On the following day these opaque lines have increased somewhat in length, whilst at the same time the middle portion of the opacity has been transformed into a shallow ulcer. Under favorable circumstances, no further increase in size takes place, the remaining opaque epithelium is thrown off and reparation begins to be completed only after several weeks. But not infrequently the ulcer continues for days and even weeks to grow slowly in length, and at the same time sends out club-shaped,

slightly raised, grayish offshoots from its sides. In some of my cases the ulcer crept across the entire cornea, and in a few others in which several ulcers appeared simultaneously on different parts of the cornea, nearly the whole epithelial layer was eventually destroyed. The middle and inner layers of the cornea generally remain transparent throughout, but in neglected or maltreated cases an extensive star-shaped opacity of a slightly yellowish-gray tint is sometimes developed in the inner layers of the central part of the cornea. A hypopyon is but rarely seen even in the severest cases, and spontaneous perforation of the cornea did not occur in any of my cases." He mentions hyperæmia of the iris. No change in T. nor in corneal sensibility detected. Severe pain in and around eye. Photophobia and lacrymation. Process of repair extremely slow—two to four months, and it differs from Horner's corneal affection occurring with herpes nasalis in diseases of the respiratory organs "in that, instead of transparent vesicles, small, opaque elevations not unlike phlyctænulæ, arranged in lines, are developed in the initial stages of the disease." In Horner's cases there is no tendency to spread.

Minor (*Am. Jour. of Med. Science*, 1881) on anæsthesia of cornea (10 cases) which is not due to pressure (œdema) or to "neuroparalysis," so-called, maintained that the sensitive vasomotor, or trophic filaments from the Gasserian ganglion, or the entire branch of the fifth nerve may be attacked. Anæsthesia was always in the cornea—in five cases in the conjunctiva. In six cases keratitis was combined with anæsthesia. Vasomotor disturbance is shown by paralysis of the vessels in the conjunctiva and iris. Dilatation of the iris vessels was sufficient to maintain an obstinate contraction of the pupil without adhesions or exudations, in spite of use of atropia. T. minus—in three cases. In five cases a history of malaria with supraorbital neuralgia. Some of these cases were plainly herpetic; in one, old herpetic scars.

Emmert, E. (*Centrbl. für Prakt. Augenheilk.*, Okt., S. 302, 1885). "Keratitis dendritica exulcerosa mycotica. Sudden violent photophobia, lacrimation and conjunctival injection, gray sub-epithelial clouding, beginning as a minute focus from which branches shoot out, or as a fine groove

which elongates, divides and sends out lateral sprouts. Through casting off the epithelium, furrow-like defects are left. Bacilli in the grooves. The disease begins spontaneously, without known cause, in May, April, August and September, i. e., in later winter or spring or fall, usually suddenly; in two or three days there is violent photophobia, lacrimation, conjunctival swelling. At the same time is seen a marginal gray subepithelial clouding, which begins as a small focus, from which branches shoot out, or as a fine streak (striation) which elongates, divides and also sends out lateral buds. Very soon the epithelium over the cloudy portions begins to be elevated and cast off, causing furrow-like grooves. The chief and secondary branches always remain fine. On account of the depth of the furrows and the intensity of the cloudiness which remains a long time, it must be conceded that the process is not only subepithelial but also attacks the anterior limiting membrane and the most superficial layers of the substantia propia." Healing always began from the part first affected. None of the usual remedies seemed to hasten the recovery. His first three cases began with an ulcer in the margin. In his case No. 3, "these branches and buds appeared as ash-gray, subepithelial, but also more deeply attacking, infiltrations which as long as they were not deprived of their epithelium, were constantly grayest in their axis, fainter toward their edges." He found bacilli in the grooves. His cases seemed by preference to attack those of tubercular diathesis. Hansen Grut saw no reason to accept this theory.

Hotz, F. C., (*Chicago Med. Jour. and Exam.*, Dec., 1891) says: "Let me assist his imagination by the aid of some familiar object in nature. Draw within the compass of the cornea the outlines of a small lanceolate leaf, with its stem at the margin and its free end in the center of the cornea; have the central vein of this leaf run in a slightly zigzag course and let the lateral veins be short. Now erase the outlines of the leaf and the skeleton of the veins is a correct representation of the specific character of a malarial ulcer of the cornea. This form of the ulcer is as pathognomonic for malaria as the mucous patches in the mouth are for syphilis. Local treatment alone is insufficient. Give your patient

quinine and you will arrest the ulceration at any stage of its progress and speedily relieve. The amelioration supervenes upon the administration of quinine (two grains every two hours), so promptly that it must be attributed to the influence of this medicine. And when the surgeon has faithfully but vainly tried for a week or two to subdue the trouble by appropriate local treatment, the sudden change following directly upon the use of quinine is so suggestive and striking as to convince even the most skeptical Thomas."

ELLETT, E. C., (*Ophth. Rec.*, 1899) did not find dendritic keratitis with the grippe, and refers to the fact that Godo, in 1880, found malaria positively in one-third of the cases. The eruption is papular instead of vesicular. He has not seen it under the age of sixteen; it occurs in males and females, whites and blacks. The history of malaria is usually intermittent; often following the first chill or any subsequent one, the eye becomes inflamed. Examination: "A minute whitish hair line, or rarely a point, on the cornea, a subepithelial exudate consisting of a row of points, pain, photophobia, bulbar injection. Kipp's supraorbitial tenderness is not marked, and is often wanting in my cases. * * Numerous white points so small that two or three may often be accommodated in the hair line's width. * * * In two or three days the epithelium ruptures over the infiltration and at the end of this time branches generally begin to form either by sprouting off from the main line or by little points forming and extending to the original branch." Appearance: "A broadening, shallow trough usually branched," base gray, cornea otherwise not disturbed nor vascular. Healing very slow. Never deep, rarely causes hypopyon, or iritis, never perforates, always leaves an opacity behind. He quotes:

HANSEN-GRUT (*Eighth International Med. Cong.*, 1884). "Superficial ulceration with a chronic course and a tendency to become serpiginous; propagation by means of buds or excrescences so that the line of demarkation of the very superficial ulcer becomes very irregular; the surrounding parts are clear and the cornea is not vascular. In no way connected with frontal herpes and not vesicular."

NOYES, H. D. (*Textbook*): "1—Lesion confined to anterior and superficial layers; ulceration (rarely much infiltra-

tion) which may become suppurative. 2—Blunted sensibility of cornea; only slight photophobia. 3—Tenderness of supraorbital nerve at its notch and pain in its radiations. 4—History of chronic malaria. Its character is mycotic and may occur without malaria; cured by local means. True malarial keratitus is not cured without quinine or arsenic."

WILDER, WM. H., (*Jour. Am. Med. Ass'n.*, Oct. 21st, 1893) reviewed the literature and pertinently asked whether all of these forms were not really different manifestations of the same disease, viz.: herpes febrilis corneæ. He believes that "the contention of the latter authors (Kipp, Hansen-Grut and Emmert) that in cases of dendritic ulceration there is no question of vesicles, does not seem to be well grounded, since most cases would probably present themselves to the surgeon only after the delicate epithelium covering the vesicles had broken down when only the minute ulcer would be seen."

Here in the Mississippi Valley we have the opportunity of seeing dendritic keratitis in all stages, both the vesicular, herpetic form and also the papular. In the clinic of the O'Fallon Dispensary, I have seen herpes, with or without facial herpes, sometimes breaking down, more often not breaking down, to form an ulcer; and in addition the distinctly infiltrative papular form, also branching, which generally becomes ulcerative. The first we designate as "herpes," the latter as "malarial keratitis."

Some authors have implied that children are never attacked. I remember one child of six years who had a typical lesion, yet the general medical clinic could find no plasmodia. However, after several weeks the patient had a distinct chill followed by fever and recovered under anti-malarial treatment.

The question presents itself, What is the nature of the lesion, and the modus operandi of its cause?

That "malarial keratitis" may be caused by other diseases than malaria is well illustrated by the following case:

April 26, 1902, at a time when several general practitioners said that this region was absolutely devoid of malaria, came F. C., 38 years old. Six days before he had had a chill with fever following it. He had been treated by Dr. Tiedemann of

this city for grippe, the diagnosis of malaria having been excluded by the absence of recurrence. From the beginning his left eye was red and uncomfortable when he attempted to read. The patient had slight catarrhal conjunctivitis R. and L. The left eye exibits a very superficial keratitis resembling by daylight a corneal abrasion without infiltration, but by focal illumination it was seen to be accompanied by an irregular, faintly outlined haziness sufficiently beaded to lead

FIG. 1.
Taken April 29th.

FIG. 2.
Taken May 1st and remains apparently unchanged until

me to suspect malaria. It showed no trace of the marked, dirty infiltration of a virulent infection. Used argent. nitrat. R. and L., atropia with cocain in castor oil O. S. and bandage. I gave him a note to Dr. Tiedemann, who repeated that the case was one of grippe.

On the following day a 2 per cent. fluorescin solution gave lesion shaped like Figure 1.

May 1st—Lesion was more clearly defined and seemed cleaner. ℞ quin. sulph. gr.iij, ferri red. gr.j, strych. sulph.

gr.$^1/_{30}$—t. i. d. ℞ Hydrarg. chlor. mit. gr.$^1/_4$—t. i. d. Used ag. $^1/_{480}$ to the everted lids; adrenalin, bisulph. of quin. (dropped) coc. et adren., A. and C. with oil and bandaged. The cornea was very anæsthetic before any drops were used. The patient is anæmic and very nervous.

FIG. 3.
May 8th.

FIG. 4.
May 15th.

FIG. 5.
Appearance at the time of the last visit.

May 2nd—Patient had severe pain over the left eyebrow at about noon yesterday. No manifest change inthe corneal lesion.

May 3rd—Severe left supraorbital pain with slight nausea. No appetite.

May 8th—Continuity of lesion begins to be interrupted;

appearance as in Figure 3. There is now visible a small, faint central haziness in the anterior portion of the substantia propria and a superficial tri-beaded infiltration in the outer lower quadrant.

May 15th—(Vid. Figure 4). No trace of original branching arrangement. Scar tissue rapidly becoming white and firm, surface still uneven (uncovered by epithelium). The central infiltration, while in the substantia propia, is quite superficial, is circular, or more accurately, roughly stellar and much less opaque in the center than in the periphery. The patient denies any opportunity of ever having contracted lues.

May 20th—Upon leaving the city for five weeks, I turned the case over to Dr. A. E. Ewing. Corneal scar still rough, but irritation has almost disappeared. Dr. Ewing treated the patient with sol. argent. nitrat. gr.j ℥j to everted lids, applying quin. bisulph. gr.xv to ℥j and ung. Hy. O. Fl. gr.iv to ℥i.

June 4th—Gave permission to remove bandage in the house.

June 15th—Still improving.

June 28th—The patient is sent back to me. Phlyctænular infiltration in upper inner border of the original scar. The patient is still very anæmic.

July 1st—Yesterday the eye felt sore. The cornea was roughened from above and in, toward the center. To-day bloodvessels begin to enter from above. The central haziness is denser. Ag. $^{1}/_{480}$, natr. biborat. sat. sol., atropiæ alk. et cocain alk. cum ol. ric., bandage.

July 2nd—Phlyctænule about well. Resumed capsules of quin. iron and strych., also calomel t. i. d.

July 15th—Doing well.

July 26th—Patient goes north. Two fine parallel bloodvessels enter the cornea above, but the scar is smooth and the cornea otherwise clear. Vision with glass $^{15}/_{38}$.

August 18th—The patient returns well. V. $^{15}/_{38}$.

Dr. Tiedemann's notes:

"April 28th. Dear Doctor: In reply to yours of Saturday, would say that Mr. C. had a slight fever and sore throat, which I diagnosticated as grippe. I gave him 5 gr.

doses of aspirin, several days later increasing the dose to 10 grs. every 4 hours, as he then complained of pains in his shoulder muscles. I made no blood examination, but will be glad to do so if you wish it."

"April 30th. I have looked diligently for malarial plasmodia in Mr. C.'s blood, but did not find any. I examined both fresh and stained specimens. The red and white cells are normal in number and relative proportion."

Six days after a malarial "chill and fever" treated with aspirin, we should expect to find plasmodia as well as recurrence.

NOYES (*Textbook*, p. 358) says: "That it (dendritic keratitis) is pathognomonic of malaria seems to me improbable, because I have seen many cases of malarial keratitis without it, and have seen one extremely typical case in which the cause certainly was not malaria but exposure to a fearful snow storm. An Irishman * * * * was brought suffering with so-called snow-blindness. I found the above streaks running through both his corneæ. This case was purely local. But true malarial keratitis, while it may have the nodular branching streaks, will not be cured without quinine or arsenic." If dendritic keratitis is assumed to be a peripheral neuritis, this case may be easily explained.

The inference that this disease is due to a terminal nerve lesion, is warranted, I believe, by the following facts:

First: Dendritic keratitis has been observed most frequently in those diseases which are especially prone to attack the nervous system—central and peripheral.

That form caused by herpes is vesicular in the beginning and is the expression of a descending neuritis caused by a lesion in the Gasserian ganglion, due to the poison of those acute fevers which, when they affect the nervous system, are more prone to attack it centrally than peripherally.

The second form, that caused by malaria, grippe, etc. (diseases which are more apt to attack the peripheral rather than the central nervous system, causing muscle paralysis, etc.) is not vesicular, but papular, in the beginning; but because it, also, is branching and follows the same irregular course as herpes, one may with reason infer that it, also, is a nerve lesion, probably purely peripheral. The extreme persistence

of the attack in those cases which do not recover immediately, points in this direction. "Nervous manifestations are usually divided into intermittent and persistent." "The pure intermittence of nervous symptoms is essentially rarer than their persistence, although, as most usual, they last only a few days." (Mannaberg on Malaria, *Nothnagel's Encyclopedia*).

Second: The lesion is possibly the result of a degeneration, perhaps inflammation, of (or along) terminal filaments of the corneal nerves. It is not necessary to assume an inflammation as the cause of the original dendritic form of opacity. The cornea becomes cloudy after death, in atrophy or from œdema. Its tissue is a connective tissue, so modified as to be transparent (during life), and its nerves are either so small as not to be noticed (if they are opaque) at their position in the refracting system, or they are also so modified as to be transparent (loss of medullary sheath). The cause of dendritic opacity may lie in a process which simply (1st) renders the subepithelial plexus opaque, at the same time giving rise to (2nd) an inflammation of the more resisting surrounding tissues with consequent breaking down into a very superficial ulcer. (The epithelium loses its support either by injury to it or by a change in Bowman's membrane). If we assume this to be the case, the foci of inflammation (i. e. the bead-like infiltrations) are probably located at the triangular branching nodes. The fine lines seen between the foci are probably either the opaque degenerated terminal bundles of the subepithelial plexus, or they are composed of leucocytes traveling along the paths of these bundles, eventually causing slight injury to them in those mild cases which recover quickly, or atrophy in those very obstinate cases which extend over months before healing even begins.

The former proposition is plausible, if we remember that nerve fibres lo-e their medullary sheaths where transparence is needed, as in the cornea and retina, or when more room is required, as in the crowding which takes place in the brain; and that, however, as soon as a non-medullated fibre degenerates, it becomes opaque and a bundle of such fibres might then become readily visible to the unaided eye.

Evidences of the origin of dendritic keratitis in a lesion of nerve terminals or fibrils are—

First: Anæsthesia, which I have rarely found wanting in these cases. This symptom originates from one of three causes—hysteria, "neuro-paralysis" and pressure œdema as seen in glaucoma. After considering the slight infiltration, as compared with that accompanying so many other corneal lesions which do not exhibit this symptom, one must regard the power of resistence of the nerve terminals to injury by pressure in these cases, as greatly diminished, to say the least, and therefore that the poison has acted directly on the terminal fibril.

Second: The shape of the lesion itself in the beginning resembles much the arrangement of the fibres in the subepithelial plexus. Then, too, the branching in our cases and in most of those published seemed to follow the directions of the nerves from the periphery either diagonally or toward the center rather than from the center toward the periphery. I do not mean that the disease does not often begin at the center, but that, what branching does occur, follows as if the base were a peripheral stem.

Emmert's cases:

1st Case—Ulcerative keratitis, at the temporal side 3 mm. in extent, from which a fine epithelial streak advances toward the center of the cornea; buds, right and left, followed, originating a furrow ulcerative keratitis.

2nd Case—A superficial infiltration with loss of epithelium at the upper corneal margin, contiguous to which began an irregular, branching, finely streaked gray infiltration, subepithelial, but also, in the anterior elastic lamina and the superficial corneal layers, extending toward the pupil.

3rd Case—A superficial ulceration at the inner upper corneal margin. Buds with furrows. Two large branches.

4th Case—Keratitis dendritica 1 mm. distant from the temporal corneal margin, with an entirely (distinct) separate branching lesion in the neighborhood of the pupil.

5th Case—A 3 mm. infiltration in lower outer portion not at edge.

6th Case—A small branching infiltration below and inward separated from the margin. Rather deep furrows.

Hotz's cases:

1st Case—Near the upper margin a small, linear abrasion with a peculiar zigzag branching line advancing toward the center.

2nd Case—A small abrasion at the upper margin, in a week extending almost across the cornea.

3rd Case—In the upper nasal section near the margin a minute "phlyctenule-like spot" from which a fine, gray linear opacity proceeded downward 1 mm., branching below.

Ellett'a cases:

1st Case—A crescentic, narrow ulcer near the inner border.

2nd Case—A fine, white, raised line diagonally across the upper half.

3rd Case—Up and out near center; grooved, gray area.

4th Case—In the upper, outer quadrant a delicate branching line.

5th Case—A typical dendritic keratitis with large, T-shaped, central lesion.

6th Case—A clear, crescentic trough of fine, white points below, and parallel to the limbus.

7th Case—A subepithelial infiltrate outward from the center, 1 mm. in diameter.

8th Case—A linear, raised infiltration in the upper temporal quadrant.

9th Case—A small, central, three-pronged ulcer.

10th Case—A delicate, raised line, curved up and parallel to the limbus at the lower, inner quadrant. Three prongs.

Wilder's cases:

1st Case—An ulcer at the upper margin, extending over the center, with buds.

2nd Case—An ulcer from above downward.

3rd Case—Superficial ulcers arranged in a branching form, confluent in places.

The question suggests itself, whether the (amœboid) movement of leucocytes is so paralyzed by pigment granules and poison that they can wander only so far and then must remain stationary to block the lymph channels.

Third: The lesion in the beginning is never below Bowman's membrane, but is directly under the epithelium.

Later, it does extend sufficiently deep to almost invariably leave a scar. Hence those cases of keratitis caused by acute malaria recover quickly under quinine, by checking the cause of degeneration before the foundation for the epithelial structure (Bowman's membrane) is injured; but if the process has extended farther, quinine does not hasten the recovery, and although the patient may have no other malarial manifestations for months, nevertheless, the ulcer will not heal. I know of no other non-virulent ulcer which is so chronic as some of these cases.

The question which naturally arises here is how far the nerve fibrils influence the epithelial cells, remaining in situ. Whether their feet are not in reality nerve terminals. Certainly, those stains which bring out most distinctly the "Kitt substance" of the epithelium are those which are known as nerve stains, e. g. osmic acid, gold chloride, Ag NO 3.

Extreme chronicity may be, first, the result of what is suggested by the foregoing, viz.: that with interference of nerve-supply (either by a primary nerve lesion or by blocking of lymph-paths along nerve fibrils—here nerve degeneration would be a result rather than cause) the nourishment of the anterior layers of the cornea is impeded; or, second, it is barely possible that the continued action of the poison causes a general degeneration of all tissues in those layers (Bowman's membrane, nerves and epithelium).

The nourishment of the cornea proper takes place through its lymph spaces which contain fixed corpuscles, leucocytes and nerves. The nutrition for Bowman's membrane and the epithelium probably follows the paths of the nerve filaments, piercing Bowman's membrane and passing between the epithelial cells. Any poison would be more likely to affect those portions of the nerves farthest removed from the source of food supply, i. e., the bundles lying in the substantia propria which contain the lymph spaces and corpuscles would be less liable to injury than their fibrils distributed to the epithelium. The toxin would enter through the same channel as the nutriment.

THE DIFFERENT FORMS OF HEREDITARY SYPHILITIC KERATITIS AND THEIR TREATMENT.*

By ETIENNE ROLLET, M.D.

Translated by Adolf Alt, M.D.

THERE are few diseases which have been written about so much as interstitial keratitis. Hutchinson's ideas on the ætiology of this affection have been accepted and it is to-day admitted that in general we have to deal with a hereditary syphilitic keratitis. Even the synonyms reflecting the older ideas on this disease have been forgotten (scrofulous, cachectic keratitis); only those based on the clinical picture, or on pathological characteristics, are still retained (diffuse, disseminated, parenchymatous, deep and interstitial keratitis). Once recognized as being of a hereditary syphilitic nature, this disease would naturally fit into the class of syphilitic affections, which are treated by specific medication.

Fournier[1] says: "The indication is to fire all the canons, that is to give simultaneously mercury and iodides; later on the treatment may be alternated by giving mercury for three months, followed by iodides for three months, and so on. It is essential to give these remedies in a full dose."

Panas[2] says: "The vigorous and prolonged employment of mercury and the iodides constitutes the basis of the treatment. Therefore, one should prescribe in turn potassium iodide, Gilbert's syrup, mercurial inunctions and subcutaneous injections of bichloride, or better, biniodide of mercury. After 25 to 30 injections it is best to stop, to again resume three or four weeks later."

According to these citations, which I could multiply easily, there is here an indication for mixed treatment which may be termed severe and intense, and we must acknowledge that even if the parents of the syphilitic child have taken no mercury, this must be insisted upon in their offspring.

We are also told that this anti-syphilitic treatment must be prolonged. Fournier says: "We must be patient, because the disease lasts long, very long." Only recently Baudry[3] wrote: "When called to a patient with parenchymatous keratitis, we must inform him and his family of the long

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duration of this disease—several months and sometimes several years."

Contrary to these writers, others especially depend on a natural cure and are, therefore, much less enthusiastic as to specific medication. Gayet[4] says: "Concerning this chapter of therapeutics, experience has taught us that we are desolately impotent. Usually, when local treatment is unsuccessful we fall back on general treatment. If iodide of potassium seems to be of some value, it surely does not bring about remarkable cures, and as to mercury, I have never thought its effect to be a certain one. In many cases I have tried mercurial treatment without obtaining any undoubted effect."

What can we say of these contradictory opinions announced by these different clinicians concerning the efficacy of specific treatment in hereditary syphilitic keratitis? The first thing that strikes us is, that the practitioners who have the greatest confidence in the medication tell us beforehand that its action is very, very slow.

Is it not really classic, having prescribed a specific treatment for diagnostic purposes, to give it up after twice seven years, because its dissolving action is seen to be nil after the lapse of this time? Clearly, we have here no ordinary tertiary lesions to deal with which are easily dispelled.

It is my opinion that the authors I have cited, having treated various forms of keratitis of different origin by means of one and the same medication, must necessarily have obtained varying results. Neither do I believe that there is but one keratitis, but that there are several kinds of hereditary syphilitic keratitis. The future will teach us whether their clinical and pathological symptoms are distinct; at any rate I believe from now on that their internal treatment is absolutely not the same. It is true, as is admitted and as I have often been able to observe, that children, adolescents and even persons in adult life, attacked by interstitial keratitis very often come from syphilitic stock, but we forget to make distinctions. Thus, I have observed especially syphilis in the father, less frequently syphilis in the mother or in both parents. In summing up, I want to establish the following classification: 1, The affection of the cornea is of some non-specific origin in the descendant of a cured syphilitic.

2, The keratitis is part of a hereditary syphilis with malnutrition. 3, Keratitis in a case of virulent hereditary syphilis. Of course, I do not speak of the interstitial keratitis which is observed in acquired syphilis.

1. In the first case the father usually acknowledges having had syphilis six, eight, twelve or more years before marrying, before the birth of the child. This syphilis was mild, well treated, perfectly cured. At the age of eight or twelve years, the child has an interstitial keratitis without any blemish in the skin, bones or teeth. I believe such a keratitis can be an ordinary one and need not be referred to the former syphilis of the father, and that it falls under the category of those forms of interstitial keratitis, with the same clinical aspect, which we encounter sometimes in the descendants of alcoholics, of tuberculous parents and of individuals who absolutely never were syphilitic.

2. This is a keratitis in a hereditary syphilis with malnutrition. In young, and oftener in adolescent patients, such late manifestations of hereditary syphilis are found in the teeth or the nose; in others a hyperostasis of the tibia is found, or other dystrophies which are in no way influenced by a mixed treatment. An important point is that such a patient is never contagious; as he has had no secondary affections, he is not immune and may contract a syphilitic chancre, as many observations have convinced me.

3. This is the keratitis of a virulent hereditary syphilis. This is more scarce, since the individual often succumbs (70 to 80 per cent.) to the specific lesions which are so murderous in early infancy.[5] This keratitis attacks an individual who has had this precocious hereditary syphilis at the nursing period. I think this keratitis of a virulent type appears earlier than the one due to mal-nutrition. I am just now treating such a case in my service at the Hôtel Dieu. It is in a girl five years of age, both of whose parents had syphilis, who, according to the classic rule, herself had undoubted syphilitic symptoms (mucous and cutaneous syphilides), and to-day she suffers from the affection which we are here considering. In a case of keratitis from virulent syphilis, the subject had the contagion at birth and may present at the same time secondary affections and different sclero-gummous

and dystrophic lesions. Although such a patient might transmit a chancre, he never had one nor will he ever have one.

If these principles regarding the multiple origin of interstitial keratitis in the descendants of syphilitics are accepted, it is easily understood that specific treatment can produce happy results only when administered in a subject of virulent hereditary syphilis; that it will remain ineffective when prescribed in a dystrophic case or the case of the descendant of a cured syphilitic. That is exactly what I have observed, and from my practice I think that specific treatment is rarely indicated, because it is applicable only in the virulent cases, the number of which is restricted. In the other cases the internal treatment should be of a tonic and non-specific kind.

Finally, whatever may be the nature of the interstitial keratitis, local treatment, which is not given any prominence by the authors here cited, gives remarkable results when applied when the disease is established, and still better while the disease is at its beginning. In order to treat an interstitial keratitis it is by far the best to employ subconjunctival injections. For a long time biniodide of mercury[6] and methylene blue[7] have given me entire satisfaction. The blue is very diffusible and causes no pain. It can be injected every two days, the needle being inserted in front of the insertions of the four recti muscles. The oleate of biniodide of mercury is of a very energetic action; the irritation caused by it is insignficant, contrary to that of other mercurial salts. Through experiments made on rabbits, with the assistance of Mr. Dubreuil, with concentrated solutions of methylene blue, I have been able to see that when injected under the conjunctiva the blue was diffused into the cornea from the periphery to the center in a progressively decreasing dilution due to the interstitial fluids. The subconjunctival injections act, as it is said, by modifying the diapedesis or by revulsion; and I want to add to this the true mechanical and a microbicidal action of the fluid which penetrates into the corneal canals. Its modifying power is the greater since the lesion it is to combat lies in the periphery of the cornea. I do not dwell any longer on this subconjunctival medication, nor on the cases of keratitis which I have treated upon. With regard

to this subject the reader is referred to the thesis of my pupil, Dargein.[8]

My desire was to show that the so-called keratitis due to hereditary syphilis can be cured quite rapidly by a local treatment, thus saving the children or adolescents affected by this malady, from a general treatment of iodides or mercury, which, I think I have successfully shown, is perfectly useless in the majority of the cases.

[1] La syphilis héréditaire tardive, Paris, 1886, p. 205.
[2] Traité des maladies des yeux, Paris, 1894, vol. i, p. 247.
[3] Baudry, Le Nord Médical, June, 1903.
[4] Gayet, Eléments d'Ophtalmologie, Paris, 1893, p. 239.
[5] E. Rollet, La syphilis des nourrissons et des nourrices au point de vue médico-légale, Arch. d'Anthropol. Crim., June, 1894.
[6] E. Rollet, Des injections sous-conjonctionales de biiodure de mercure, Lyon Médical, Feb., 1899.
[7] E. Rollet, Des injections sous-conjonctivales de blue de méthylène, Lyon Médical, Feb., 1901.
[8] Dargein, Traitement des Keratites par les injections sous-conjonctivales (blue de méthylène, biiodure de mercure), Thèse de Lyon, Jan. 12, 1904.

A CASE OF OVER-CORRECTION OF CONVERGENT SQUINT WITHOUT OPERATION.

By SAMUEL THEOBALD, M.D.

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CASES of over-correction of convergent squint following operative procedures, though less common now-a-days than they once were, are not so rare as to make them noteworthy; but instances of a well-marked inward squint being converted into an outward squint, simply through the influence of glasses, are certainly very uncommon. And so it has seemed to me worth while to place upon record a case of this character which has recently come under my observation.

Master E., a lad, twelve years of age, was first seen January 30, 1901, when he was brought to my office for advice regarding the condition of his eyes. There was a fixed and marked convergent squint of the right eye, and there was given a history of asthenopia. The squinting eye was found to be descidedly amblyopic ($V = {}^{20}/_{100}$), and the sight of this

eye could be improved but little by glasses. A test under hyoscyamine, made the following day and repeated the day after, showed the total refractive error and the vision of each eye to be as follows:

L. eye + 3.25 s.⌒ +.50 c. 85° V⌒ $^{20}/_{25}$
R. eye + 3.50 s.⌒ +.50 c. 95° V⌒ $^{20}/_{90}$ —

In accordance with my usual practice, I should have urged tenotomy, and should not have thought it worth while to attempt a correction of the squint by glasses alone; but, as a year previously I had succeeded in correcting with glasses a periodic convergent squint in a younger sister of the patient, I concluded to try the same method with him. Accordingly, after the effect of the cycloplegic had passed off, the following glasses, which somewhat over-corrected the manifest hypermetropia, were prescribed for constant use:

L. eye + 2.50 s.⌒ +.50 c. 85° ⌒ prism 2°, base out.
R. eye + 2.75 s.⌒ +.50 c. 95° ⌒ prism 2°, base out.

The patient was seen about a week after getting these glasses, when there was no appreciable change in the position of the squinting eye. Nearly two years elapsed before his next visit. He had worn his glasses systematically, and, in spite of the fact that the left lens had become misplaced, the base of the prism having been turned toward the nose and the axis of the cylinder to 95°, he had been free from asthenopia. As shown by the cover test, there was still, with the glasses as he had been wearing them, a slight residual convergent squint of the right eye, the vision of which was unchanged.

As a result of a re-examination of his refraction, this correction, for constant use, was prescribed, Jan. 5, 1903:

L. eye + 2.50 s.⌒ +.75 c. 75°
R. eye + 3.50 s.⌒ prism 3°, base out.

He was not seen again, except to show me his glasses a few days after they were ordered, until December 29, 1903, nearly twelve months afterwards. He had worn the glasses constantly, and had been free from asthenopia. There was now, with the glasses on—and this was confirmed by the cover test—an evident *divergent* squint of the right eye. In view of this, his lenses were changed as follows:

L. eye + 2.25 s. ⌒ +.75 c. 80°
R. eye + 3.25 s.

These about corrected the manifest refractive error in each eye, and gave for the left eye V = $^{20}/_{25}$ —, and for the right eye V = $^{20}/_{60}$ —, indicating a slight improvement in the sight of this eye. With this correction, the right eye was disposed still to squint outward, though at times there seemed to be binocular fixation in distant vision. After ten days the right eye was found to be still squinting outward, and, what was more remarkable, this outward squint persisted even when the glasses were removed. A month later (Feb. 5, 1904), there was no change in the position of the eyes in distant vision, but the cover test seemed to indicate a disposition to binocular fixation in near vision. He had, besides, suffered somewhat with headache after reading, which I thought due, probably, to an effort to establish binocular fixation. March 9th, with his glasses on, there was still a divergent squint of the right eye in distant vision and, most of the time, in near vision, though occasionally there seemed to be binocular fixation in near, especially in reading.

By the vertical diplopia test, and with a red glass before the left eye, there was at 20′ an "exophoria," if it could be called so, of 10° to 11°, and at 12″ of 4°. Without glasses, the right eye, as before, squinted outward in distant vision; and when the left eye was covered, and he fixed with the right eye, the squint shifted, in the usual manner, to the covered eye.

This case seems to me to possess several features of interest. In the first place, it shows very strikingly how great an influence glasses exert at times over the position of a convergently squinting eye. In the next place, it illustrates the pronounced indisposition to binocular vision which, as we all have had occasion to observe, exists in certain strabismic individuals. In some instances the difficulty in establishing binocular fixation, whether by the aid of glasses alone or by operation supplemented by glasses, is doubtless due to the amblyopia of the deviating eye; in others, to a slight uncorrected upward or downward squint of this eye; but in not a few it can be attributed only to a lack of disposition to fuse mentally images formed upon corresponding retinal points. This last

mentioned condition, probably, was a factor in my case; at all events, there was no vertical deviation, and the amblyopia, though marked, was not excessive. Another noteworthy feature was the persistence of the upward squint when the quite strong glasses which the patient was wearing were removed. This would not have been so remarkable if he had made no attempt to obtain clear vision by accommodative effort; but even when he was induced to distinguish small letters at 20′, no change in the position of the deviating eye could be observed. Probably a change in this regard would have occurred had the glasses been left off for several days. Should the squint not disappear within the next few weeks, it is my intention to reduce further the strength of the spherical correction; and I am not without hope that in this way I shall be able eventually to secure binocular fixation.

As an addendum, it may be of interest to mention that the periodic squint in the case of the patient's sister, who when first seen was but five and a half years of age, was corrected by the following glasses:

L. eye + 4.25 s.⌒ prism 2°, base out.
R. eye + 4.25 s.⌒ prism 2°, base out.

These were subsequently changed to

L. eye + 4.25 s.
R. eye + 4.50 s.

with which she has, at the present time, not only comfortable binocular vision, but practically normal muscle balance. A favorable feature in her case was the existence of acute vision in the periodically squinting eye.

RUBBER-DAM DRESSING AFTER ENUCLEATION.

By L. R. CULBERTSON, M.D.
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THE removal of the first dressing after enucleation, when gauze is used, is nearly always very painful, and anything that prevents this is a great boon to such patients. Even when the gauze strips are saturated with vaseline, they will stick and cause pain in removing. Recently in reading of McKernon's method of spreading rubber dam over the

surface of the wound after a mastoid operation to prevent adhesion of gauze at the first dressing, I thought how useful it would be after enucleation. At the first opportunity I tried it and it was a great success. A piece of medium weight rubber-dam was cut round. Then I cut several very small holes in it from $^1/_{16}$ to $^1/_8$ inch in diameter (these to go in the back of the cavity) so as to allow any blood or pus to flow through and get on the gauze. The dam was then pushed back in the cavity and the iodoform gauze pushed in upon it, filling up the cavity.

The dam should be large enough to cover the conjunctiva of the lids. Before putting in the dam all hæmorrhage should be arrested with hot water or adrenalin (1—5000). Before the operation the dam should stand for an hour in 1—1000 corrosive sublimate solution. Just before dressing it should be rinsed in sterile water and dried in a sterile towel. It can be used at the second dressing, but the pressure used in introducing it may be painful to some. The dressing may be removed on the second or third day after the operation.

ON REMOVAL OF THE CRYSTALLINE LENS IN HIGH DEGREE OF MYOPIA, AS ILLUSTRATED IN SIXTY CASES.*

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FOUR years ago I contributed a paper to the discussion on the operative treatment of high myopia before the Ophthalmological Society, and which is published in the *Transactions* of that Society. I there dealt with twenty operations. Since then I have increased the number in which I have removed the crystalline lens to about sixty, and it is the experience thus gained that I propose bringing before your notice to-day.

The deplorable results associated not infrequently with high degrees of short sight are well known. The inconveniences attendant on the use of the necessary powerful

*Prepared for a meeting of the Yorkshire Branch of the British Medical Association.

glasses are great. Without their aid, those unfortunately afflicted with high myopia are well-nigh blind, and not infrequently having removed and deposited their glasses they are with difficulty able to find them again. To relieve such cases as here depicted the removal of the crystalline lens has been suggested and performed with success in this country and abroad during the last few years.

Donders regarded the cure of myopia as belonging to the *pia vota*. Before his day the removal of the lens had been suggested, but, writing in 1864, Donders treated the proposal with ridicule, and insisted that such a momentous undertaking "would exhibit culpable rashness." The forty years which have elapsed since Donders wrote these words have witnessed many changes in ophthalmic surgery. Asepticism, which has enabled so many operations to be accomplished with safety in general surgery, has extended its benefits, if in a less degree, to the operative surgery of the eye, and the attendant risks in careful and skillful hands are much less now than formerly.

Within defined limits the removal of the crystalline lens for high degrees of myopia may now, I believe, be regarded as a recognized and valuable method of treatment. By its means the myopia will be either entirely obliterated and the eye become emmetropic or a varying degree of low myopia or a low degree of hypermetropia will result, according to the original degree of shortsight.

A consideration of my cases will bring into view several points which should be noticed. The number of patients operated upon has been 40, the number of eyes 59. It may often be well to be content with only treating one eye, and my rule has been to take the worst eye. Frequently, however, my experience is that after having undergone operation on one eye the patient has been so pleased with the result that urgent application is made for the other to be similarly treated. Domestic servants are numbered among those who have returned after one eye has been operated upon and have been desirous to have the second one done. I am always more willing to accede to such wishes in young adults. In one instance, however, a married woman, Mrs. D., aged 48, I treated both eyes four years ago with most gratifying results.

The number of operations reviewed in this article is 59. There were 40 patients; in 21 only one eye was treated and in 19 both eyes were operated upon. Of the 40 patients, 11 were males and 29 were females. Of those who underwent operation in both eyes 13 were females and 6 were males.

One patient was aged 48, but this was the only instance over 40. Of the others, one was 37, another 35, and two 32. Those between 20 and 30 numbered eighteen, as follows: three aged 20, one aged 29, four aged 28, two aged 27, three aged 26, one aged 25, one aged 23, two aged 22, and one aged 21. Under 20 there were seventeen, namely: two aged 19, five aged 18, one aged 17, three aged 15, one aged 14, one aged 13, two aged 11, one aged 10, and one aged 9.

Of the instances in which the degree of myopia is accurately recorded, I find that 28 operations were performed on eyes with 20 D and above, namely, 15 for 20 D, 1 for 21 D, 2 for 22 D, 1 for 23 D, 1 for 24 D, 2 for 25 D, 3 for 26 D, 1 for 27 D, and 2 for 28 D. The lowest degree for which removal of the lens was performed was 10 D, and this in one instance only. There was one eye for 11 D, 2 for 12 D, 2 for 13 D, 3 for 14 D, 1 for 15 D, 3 for 16 D, 10 for 18 D, and 1 for 19 D. The number, therefore, between the eyes of 10 and 19 D inclusive was 24. It will thus be seen that practically all the operations have been performed on eyes having 12 D and more degrees of myopia, the two exceptions being 10 D and 11 D respectively. Moreover, the greatest number of operations have been for 18 D and upwards, 18 D being the degree in 10 eyes and 20 D in 15.

If the operation be limited, as I believe it should be, usually, to cases of myopia of 14 D and upwards the total number of suitable cases presenting themselves must always be a comparatively small one. In this connection I have taken from the casebooks of my private practice the degrees of myopia in 3,162 consecutive patients or 6,324 eyes, a number which should give a fair idea of the proportion of the different degrees of myopia. The cases were roughly divided into those above 10 D and those of 10 D and under. If the division had been made at a higher degree of myopia, which, strictly speaking, it should have been, as, save in exceptional instances, the operation would not be performed for less than 13 D or 14 D, the total number of instances suitable

for operation as far as the degree of myopia is concerned would be decidedly less.

Of these 3,162 patients I find that in 1,159 the degree of myopia was equal in each eye. In 2,003 the eyes were unequal in refraction; that is to say, that while myopia was present in one eye it was so to a higher degree in one eye than the other, or in some the second eye may have been emmetropic, or hypermetropic, but the greater number would be myopic. Of these 2,003 eyes the degree of myopia was higher in the right eye in 1,029, and in the left in 974. Of the total number of eyes under review, namely, 6,324, there were 320 in which the degree was more than 10 D, or a trifle over 5 per cent. The record of my infirmary patients would, I expect, show a larger percentage of the high degrees of myopia, as those with the low degrees less frequently seek advice there than is the case in private practice.

Extraction was deemed unsuitable, and in no instance has it been adopted. In each case the transparent lens has been needled, and at the end of a week, sometimes more or less, when the lens substance has become sufficiently broken up, an incision with a broad needle has been made a little distance inside the periphery of the cornea, and the softened lens has been allowed to escape, or has been coaxed out with the aid of a curette. The pupil has previously been dilated with atropine, and by keeping the situation of the corneal wound inside the limit to which it dilates, the danger of entanglement of the iris in the wound is materially lessened. This operation is similar, of course, to that frequently performed for zonular and other forms of cataract. In all cases a watch must be kept for increased tension, which is liable accasionally to set up by the swelling of the lens. In a few of my cases glaucoma has in this way supervened. Removal of the broken-up lens was, however, promptly performed, and was followed by immediate subsidence of the pain and sickness, and with excellent visual results.

When operating, endeavor is, of course, made to remove as much of the softened lens matter as will escape without undue pressure on the globe. What remains, however, will undergo absorption, but a further needling may be required. Needling may also be necessary for opaque capsule and it is therefore important to bear in mind that treatment in some

cases may be somewhat prolonged. In one of my early instances I was perhaps hardly sufficiently alive to the importance of this and operated rather against time, as the patient was leaving England.

If due regard be paid to asepsis and the use of sublimate or some other antiseptic solution which are essential to eye operations, the dangers attendant on this operation are trifling. The introduction of any suction apparatus is altogether unnecessary, for by gentle coaxing the softened lens matter readily comes away through the corneal wound. It is desirable, however, that after the needling operation the patient be kept under close observation, for, as mention has already been made, in some instances the swollen lens is apt to induce increased tension as signified by pain and sickness, and the immediate performance of the second operation for the removal of the broken-up lens will be indicated. A free opening of the capsule is practiced, because it is held that by permitting the broken-up lens to escape into the anterior chamber increased tension is less likely to be induced. This is not, however, the only period that it is necessary to be on one's guard against the onset of increased tension.

In a certain number of cases after the usual operative measures have been completed and the pupil is free, or nearly so, of lens matter, increased tension may occur. In these instances atropine has very properly been used to maintain a dilated pupil whilst the remnants of the lens were undergoing absorption. The atropine may be discontinued and eserine substituted, but it will, in my experience, fail frequently to overcome the glaucomatous condition. The best plan is to tap the anterior chamber, and to repeat if necessary the paracentesis. In the few instances that increased tension has arisen in the circumstances just now mentioned, paracentesis has acted well, and the results in those cases have been among my best.

The operation performed in my cases is similar to that practiced for lamellar cataract, and the dangers are the same. A bead of vitreous may present at the wound, but this should very rarely occur if care has been taken to limit the tearing of the capsule to the anterior capsule, which should readily be done, and also if too great pressure on the globe is avoided when the lens is coaxed out by the curette.

My practice has been to indicate to a patient the advantages which may be gained by operative measures, but at the same time to point out that to achieve the benefits he or she must be prepared to face the risks which, however, are not great. The decision is left to the patient.

Some authors have alluded to the danger of detachment of the retina resulting from removal of the crystalline lens for high degree of myopia. I have no experience of extraction of the lens in these cases, and cannot therefore say whether it is a danger by that method or not. All my cases have been treated in the manner I have described, and certainly detachment of retina is not a danger of moment in my experience. I recollect only two instances; one was mentioned at some length in my paper before the Ophthalmological Society.

Case I.—The patient was a young woman aged 25. The lens was needled, and subsequently evacuated in November, 1897. The result up to May, 1898, was excellent, myopia of 22 D had gone down to 2 D, and vision at this time was $^{6}/_{24}$. Shortly after this, however, owing to the serious illness of a near relative, the patient had very arduous night and day nursing thrown upon her. Vision rapidly deteriorated, and on June 8th it was reduced to $^{3}/_{60}$. Besides some films in the vitreous, no gross changes were noticeable in the eye. Vision remained at this point or a little worse for some months, but she had not been under examination for some time. It should be mentioned that when the lens matter was evacuated a small amount of vitreous escaped.

Case II.—The second was that of a domestic servant. Both eyes had been operated on with excellent results. A year or more afterwards detachment of retina occurred in one eye. At this time she was in very feeble health. The other eye has remained good as far as I have heard.

These are the only instances of which I have either record or recollection in the 59 eyes which have been operated upon, and many of my cases have been under observation more or less for several years. Myopic eyes—especially the high degrees which are dealt with in this paper—are, it is well known, prone to detached retina. They are cases in which a sword of Damocles is always hanging over the heads. Even, therefore, if the percentage was much larger, as it has

been in some published records of instances in which detached retina has occurred after varying periods subsequent to the operation, it could hardly be regarded as discrediting the operative methods here advocated.

The results of a successful operation to the patient will be that vision is as good or better without glasses for distance as it was previously with their assistance. The myopia will have disappeared or be present only in low degree, or there may be weak hypermetropia, according to the amount of myopia originally existing. It would be very unusual for a patient not to express his or her gratification with the change that has been brought about. There are few subjects in ophthalmic surgery that elicit such expressions of gratitude as do these operations for the relief of myopia.

I give here an extract from a letter received from a patient operated upon some few months since. She was a teacher, aged 28, and the degree of myopia in each eye was 28 D. The right eye only was treated. Before operation, with correction with —28 D, V=$^{6}/_{18}$; after operation, without the aid of glasses, V=$^{6}/_{12}$.

"The benefits received from the operations are so numerous that I hardly know how to specify them. The operations, of course, to me appear nothing short of a miracle. The fact that I had worn exceedingly strong glasses for twenty-two or twenty-three years emphasizes this. Prior to the operation objects a few yards distant were quite unrecognizable; clear vision was only obtainable an inch or two from the eye. The uncertainty of locating the outline of objects caused me great trouble, inconvenience and, at times, even fear. For instance, in crossing the road the edge of the pavement was always uncertain. The same may be said of steps to vehicles, stairs, etc. Nothing of that uncomfortable uncertainty remains, all things are now clear. I see clearly, distinctly, and have quite a long range of vision; not only that, but the continual tired, aching feeling, and fearfully hot sensation have entirely gone; and it is saying a great deal when I say that whereas for many years my eyes have been an ever-present feeling, I now sometimes forget that I have eyes, so very natural and painless are they."

[Since this article was written I have operated on four additional cases, all successful; three females, aged 26, 15, 9, one boy aged 10; the degree of myopia of each in this order being 18 D, 14 D, 18 D, and 15 D.]

ABSTRACTS FROM MEDICAL LITERATURE.

By W. A. SHOEMAKER, M.D.
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THE PHYSIOLOGY OF THE SYMPATHETIC IN RELATION TO THE EYE.

G. E. De Schweinitz (*Jour. A. M. A.*, Jan. 30,) from a review of the entire literature of the subject, and from the opinions of the various authors quoted, offers the following conclusions:

1. Athough lacrimal secretion may be caused by excitation of the sympathetic, and increased lacrimation by section of the cervical sympathetic or removal of the superior cervical ganglion, the sympathetic itself should not be considered the nerve of secretion for the lacrimal gland.

2. Dilatation of the pupil is probably caused by contraction of a set of radially arranged muscular or contractile fibers, the so-called dilatator pupillæ, which is supplied by the sympathetic, and by inhibition of the sphincter of the iris. The dilating impulse transmitted to the iris passes through the cervical sympathetic and in general terms along the mydriatic tract of the pupil, which proceeds from a center in the medulla as far as the second dorsal nerve, follows its communicating branch to the cervical sympathetic, and arrives at the internal carotid plexus, from which point it passes to the nasociliary branches of the nasal nerve, which as the long ciliary nerves supply the muscular tissue of the iris.

3. Although experimental and clinical evidence favors the presence of a center situated between the spinal cord and the exits of the sixth cervical and fourth dorsal nerves, to which Budge relegated the origin of the pupil-dilating fibres of the sympathetic, its existence has not been definitely proven.

4. Although the nature of the ciliary ganglion has not been positively determined in any one species of animal, and, although it differs greatly in different species, the weight of evidence is in favor of the ganglion belonging to the sympathetic system, at least in so far as man is concerned. The root fibres which belong to the oculomotor end in the ciliary

ganglion, where a new neuron begins for the fibers which pass to the ciliary muscle and the sphincter of the pupil, i. e., the oculomotor does not act directly on the sphincter of the pupil, but only in association with the ciliary ganglion. There is a certain amount of evidence that this ganglion is related to the pupil movements in the form of a center, and it probably contains cells which are active in the sensibility of the cornea, but lesions of the ganglion itself, although they have been considered by Grosz to be the basal cause of true neuroparalytic keratitis, have not been proved to sustain this position by experiments, inasmuch as trophic changes have not been observed after extirpation of the ganglion. Removal of the ganglion has little or no influence on intraocular tension, and its excision is not a rational procedure for the relief of glaucoma.

5. There is no satisfying evidence that the sympathetic is related to the function of accommodation, and it has not been proved that the sympathetic has any power in causing negative accommodation, nor has it been demonstrated that alterations in refraction noted after stimulation of the sympathetic are due to actual change in the lens.

6. Electrical stimulation of the cervical sympathetic produces at first an increase and later a decrease of intraocular tension, the increase being probably due to an effect on the vessels of the eye. Slow-acting, mechanically produced irritation of the symathetic causes a rise of tension, which, according to Ladoto, is independent of dilatation or constriction of the blood vessels, and also independent of the state of the pupil. Section of the sympathetic, or extirpation of the sympathetic ganglion, is followed by a fall of intraocular tension, which probably depends on vascular and, perhaps, muscular changes. The lowering of tension is more decided after excision of the ganglion than after section of the sympathetic cords, but in either case the effect is a temporary one, and may not last more than a few days, and sometimes disappears within a few hours.

7. Electrical stimulation of the cervical sympathetic produces on the side stimulated a dilatation of the pupil as a result of contraction of the dilatator pupillæ, associated, perhaps, with an inhibition of the sphincter. At the same time there may occur on the opposite side a contraction of the

pupil, which either depends on the consensual pupil reflex, or represents a reflex transmitted through the sympathetic fibres joining the cranial nerves in the region of the cavernous sinus.

8. Electrical stimulation of the cervical sympathetic causes retraction of the nictitating membrane and proptosis, owing to the action transmitted to the unstriped muscular fiber. In contrast to the general rule, irritation of the sympathetic in rabbits causes a retraction of the eyeball in the orbit, which has been attributed by Heese to a contraction of the orbital vessels and the anemia which this causes.

9. Electrical stimulation of the cervical sympathetic is followed by a contraction of the blood vessels of the conjunctiva and of the iris, and perhaps by alteration in the caliber of the vessels of the retina, although observations on the last-named phenomenon have been extremely contradictory.

10. Stimulation of certain areas of the brain cortex causes dilatation of the pupil, associated, if the cervical sympathetics are intact, with all the symptoms of stimulation of the cervical sympathetic. Division of the sympathetic stops the other symptoms, but not the dilatation of the pupil, which is supposed to be due to inhibition of the tonic action of the third nerve (Parsons).

11. Sympathectomy or gangliectomy causes the following effects: Myosis, narrowing of the palpebral aperture, projection of the nictitating membrane, retraction of the globe of the eye, hyperemia of the vessels of the conjunctiva, increased lacrimal secretion, diminished intraocular tension, certain ophthalmoscopic and microscopic lesions in the eyeground, and possibly trophic disturbances.

12. The symptoms of sympathetic section or paralysis lessen after a time, myosis being the most permanent, lasting sometimes for years. The degree of permanence, however, of the paralytic phenomena varies much in different animals.

13. Myosis is greater after excision of the cervical sympathetic cord, because it is probable that a certain tone is exercised by the ganglion; that is, that it has a different, and, as it were, a stronger action on the eye than the nerve trunk itself (Levinsohn).

14. All the phenomena of paralysis of the sympathetic

nerve, especially the contraction of the pupil, which follow extirpation of the superior cervical ganglion gradually become less marked and may disappear or even give place to the opposite condition, especially if the animal is anesthetized or subjected to sensory or emotional stimuli. In other words, extirpation of the upper cervical ganglion causes the symptoms of sympathetic paralysis which may disappear and give place to the signs of sympathetic excitation. Such paradoxical pupillary dilatation may depend on degenerative processes in the post-cellular nerves of the ganglion (Langendorff).

15. The myotic pupil, which follows sympathectomy or gangliectomy, responds to light stimulus, is still further contracted by eserin, and may be dilated by atropin. It is uninfluenced by cocain, which, however, may exercise its influence in widening the contracted palpebral fissure.

16 A considerable excision of the sympathetic must be made in order to prevent a rapid regeneration.

17. Narrowing of the palpebral fissure, ptosis sympathica, and enophthalmos are probably due to relaxation of Müller's muscle, aided, perhaps, by atrophy of the orbital fat. It has not been proved, although it has been asserted, that there is an actual reduction in the size of the globe, that is, a true microphthalmos, under these circumstances.

18. Sympathectomy or gangliectomy may cause increased vascularization of the eyeground, perhaps hæmorrhages in the ciliary body and ciliary processes, and alteration in the retinal ganglion cells.

19. Puncture of the restiform body produces just the opposite effects of destruction of the sympathetic (Dupuy).

20. Nicotin paralyses the activity of ganglionic nerve cells in the sympathetic. Cocain dilates the pupil by stimulating the mydriatic nerve endings in the iris. Atropin dilates the pupil, partly by a paralytic action on the oculomotor endings of the sphincter, and, perhaps, by a stimulant action on the sympathetic nerve fibers, or more likely, by causing a general paralysis of the unstriped pupillary muscle. Instillations of adrenalin, ordinarily inactive in causing dilatation of the pupil, become exceedingly active when the sympathetic is cut or the ganglion removed, and cause under these circumstances marked dilatation of the pupil.